Central Hypotensive Action of Propranolol Is Mediated By Opioidergic But Not Beta Adrenergic Receptors in the Rostral Ventrolateral Medulla.

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Centrally-acting antihypertensive drugs have been shown to produce their hypotensive actions through an interaction with catecholaminergic and opioidergic neuronal systems. The rostral ventrolateral medulla (RVLM) represents a major site of descending input on sympathetic outflow where these two systems converge. The present study evaluated the contribution of central beta blockade and opioidergic neuronal systems as mechanisms for the hypotensive actions. Male spontaneously hypertensive rats were anesthetized with pentobarbital, ventilated artificially and instrumented for recording arterial pressure. Bilateral microinjection of l-propranolol (O.25 nmoles) into the RVLM resulted in a significant reduction in arterial pressure which was significantly attenuated by pretreatment with naloxone (bilateral RVLM injection, 20 nmoles). A similar reduction in arterial pressure was observed following RVLM administration of d-propranolol (0.25 nmoles) which was also attenuated by naloxone pretreatment. Unlike l-propranolol, the d-isomer does not have beta blocking activity. The results of this study implicate a role for opioidergic neuronal systems in the RVLM in the hypotensive actions of beta blocking drugs. Since d-propranolol produced an analogous response to the l-isomer, this would argue against a role for central beta adrenoceptors in the hypotensive actions of these drugs.